Questions and Answers on
Air Toxics,
the U.S. Environmental Protection Agency's National Air Toxics Assessment (NATA)
and Massachusetts Allowable Ambient Levels (AALs)

What are toxic air pollutants?

Toxic air pollutants are known or suspected to cause cancer or other serious health effects, such as reproductive effects or birth defects, or to cause adverse environmental effects. Generally, the toxic air pollutants of greatest concern are those that are released to the air in amounts large enough to create an unacceptably high risk to human health, and to which many people may be exposed. Title III of the 1990 Clean Air Act Amendments identifies 188 hazardous air pollutants (HAP) likely to have the greatest impact on ambient air quality and human health.

What are some examples of air toxics?

Toxic air pollutants exist as particles, liquid aerosols, or gases. Examples of gaseous toxic air pollutants include: benzene, toluene, and xylenes, which are found in gasoline; perchloroethylene, which is used in the dry cleaning industry; and methylene chloride, which is routinely used as an industrial solvent. Examples of air toxics that are typically particles or associated with particles include: heavy metals such as cadmium, mercury, chromium, and lead compounds; and semivolatile organic compounds, such as polycyclic aromatic hydrocarbons (PAHs), which are generally emitted during waste and fossil fuel combustion.

What are the effects of toxic air pollutants?

Toxic air pollutants can have serious effects on human health and the environment. Human exposure to these pollutants can result in short-term and long-term effects. Many factors can affect how different toxic air pollutants affect human health, including the quantity to which a person is exposed, the duration and frequency of the exposure, the toxicity of the pollutant, and the person's overall health and level of resistance or susceptibility.

Short-term exposures can produce effects such as eye irritation, nausea, or difficulty in breathing. Long-term exposures to some air toxics may result in damage to the respiratory or nervous systems, birth defects, and reproductive effects. In addition, toxic air pollutants can have indirect effects on human health through deposition onto soil or into lakes and streams, potentially affecting ecological systems. Eventually, human health may be affected through consumption of contaminated food.

What standards are used to assess air quality?

The federal government has ambient air quality standards for 6 chemicals (ozone, oxides of nitrogen and sulfur, particulate matter, lead, and carbon monoxide). In addition, the federal government and some states have identified toxicity values for many other chemicals, including those referred to as "air toxics," that are used to assess air quality. These toxicity values are health protective concentrations below which there is minimal likelihood of producing adverse health effects. The Massachusetts Department of Environmental Protections (DEP) has established Allowable Ambient Limits (AALs) as its health protective guidelines; they are available at: http://www.mass.gov/dep/ors/files/aallist.pdf

What is NATA?

The 1990 Clean Air Act Amendments gave the U.S. Environmental Protection Agency (EPA) the authority to reduce emissions of 188 hazardous air pollutants (HAPs), or air toxics. The list includes pollutants that are known or suspected to cause cancer and/or other serious health effects, such as birth defects or reproductive damage. EPA conducts a National Air Toxics Assessment (NATA) as a part of its national air toxics program. The NATA process includes:

- Expanding air toxics monitoring
- Developing emission inventories
- Conducting national and local-scale air quality analyses
- Exposure modeling
- Characterizing risks associated with air toxics exposures
- Research on health and environmental effects and exposures to both ambient and indoor sources.

The NATA program helps EPA set program priorities, characterize risks, and track progress towards meetings goals.

What is the National Air Toxics Screening Assessment?

As part of the NATA program, EPA conducted a national screening-level assessment using 1996 emissions inventory data to characterize air toxics risks nationwide. This assessment helped to characterize the potential health risks associated with inhalation exposures to 33 air toxics and diesel particulate matter. These 34 air toxics were identified as priority pollutants in EPA's Integrated Urban Air Toxics Strategy. A list of the 34 selected toxics can be found at: http://www.epa.gov/ttn/atw/nata/34poll.html.

The 1996 assessment included:

- Compiling a national emissions inventory for outdoor sources of the 33 air toxics emissions and diesel particulate matter.
- Estimating 1996 air toxics ambient concentrations across the continental US.
- Estimating 1996 population exposures across the continental US.
- Characterizing potential public health risks due to inhalation of the 33 air toxics and diesel particulate matter.

What emission sources are considered in the 1996 EPA assessment?

Toxic air pollutants can be emitted from many sources, including large manufacturing facilities (e.g., chemical production plants), combustion facilities (e.g., waste incinerators), small commercial operations (e.g., dry cleaners), and on-road and non-road mobile sources such as automobiles, lawnmowers, and jet skis. Background pollutant concentrations resulting from natural sources and persistent historic emissions were also considered. Background concentrations may include contributions from past emissions, global transport, and natural sources, e.g., volcanoes.

How do the models used in the 1996 NATA work?

To estimate concentrations of toxic air pollutants in the ambient air, EPA used the Assessment System for Population Exposure Nationwide (ASPEN) model, which mathematically represents the physical processes of transport and dispersion of emissions from air pollution sources. Modeling provides a way to estimate combined impacts from one or more sources where actual measurements of the concentrations of pollutants in the ambient data are not available. Generally, the following types of information go into a model: stack height; emission rate of each contaminant; exit velocity of the contaminant; temperature of the contaminant; and meteorological data such as wind speed, direction, temperature, and atmospheric turbulence. The ASPEN model created estimates of outdoor air toxics concentrations across the U.S. measured in micrograms per cubic meter (ug/m$^{3)}$. To check the accuracy of the modeling results, the he modeled air toxics concentrations were compared to limited monitoring data, and the results were generally within two orders of magnitude. This means that, although the estimated concentrations can be considered to be within the correct range, actual concentrations may be higher or lower. The modeling results are estimated concentrations and should not be considered real, measured concentrations.

For the exposure -modeling portion of the analysis, EPA used the Hazardous Air Pollutant Exposure Model (HAPEM), which is a screening-level inhalation model. EPA used the results from the ASPEN model in the HAPEM model to provide a more realistic estimate of actual population exposure. The HAPEM model does this by accounting for time people spend indoors and in other "microenvironments" (e.g., in vehicles), patterns of movement (e.g., commuting) and activity levels to calculate human exposure.

What are the results of the ASPEN modeling with respect to Massachusetts Allowable Air Limits?

Massachusetts Allowable Air Limits (AALs) are health-based ambient air toxic guidelines that are used in permitting some stationary sources (measured in ug/m^3). These AALs are based on potential known or suspected carcinogenic and toxic health properties of individual compounds. Safety factors are incorporated into the AALs to protect sensitive people and children, and to account for other exposure pathways, like food, soil, and water. For cancer risk, AALs denote the concentration of a carcinogen associated with a one in a million excess cancer risk over a lifetime of exposure. For non-cancer benchmarks, the concentration represents the value likely to present no appreciable risk of adverse non-cancer effects with long-term continuous inhalation.

The AALs used in DEP's analysis serve as general indicators of air quality and the potential risk to public health. The comparison of predicted or monitored ambient concentrations to AALs cannot be used to predict the likelihood of a particular cancer or non-cancer effect. Exposure to chemicals above an AAL does not automatically mean that the individual will develop a cancer experience or non-cancer effect. The duration of the exposure, exposure level, and sensitivity of the individual are all factors in the development of these conditions. However, the risk of developing cancer increases with the frequency and severity of exposure.

As of August 2000, the ASPEN results for 1996 indicated that ethylene dichloride, perchloroethylene, formaldehyde, benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, methylene chloride, arsenic, and acetaldehyde were estimated to be over a MA AAL in more than one county in Massachusetts. For ethylene dichloride, chloroform, and carbon tetrachloride, these elevated levels are due to background. EPA considers background concentrations to consist of long-range transport from other geographical areas, re-suspension of historical emissions, and naturally occurring sources. In general, exceedances should be taken to indicate HAPs of potential concern, but estimated levels may vary widely from actual measurements due to the uncertainties with the modeling exercise discussed below.

The eleven toxics mentioned above are commonly found in: gasoline, dry cleaning fluids, refrigerants, fossil fuels (which are often burned to produce energy) and garbage (which is often burned to reduce volume before disposal). Please see the tables below for a more detailed list of these HAPs and their sources.

How can local, state and federal environmental and public health agencies use the results of EPA's study?

Agencies can use the modeled ambient concentrations and the exposure modeling to:

- Identify potentially important emission sources and pollutants;
- Prioritize future regulatory programs to address those pollutants and emission sources that may present the most significant threat to public health;
- Evaluate the effect of local, state, and federal air toxic control programs on reducing ambient pollutant concentrations since 1990;
- Target ambient monitoring and emissions inventory efforts.

What are the limitations and uncertainties of the results?

There is a degree of uncertainty associated with modeling estimates, because of the many assumptions used to simulate real-world conditions. The NATA results are most reliable when analyzed on a national or state scale, and are increasingly less reliable as the scale gets smaller. In addition to concerns with modeling, significant data input errors have been found in the NATA input files. Therefore, comparisons on a scale below the county level may be misleading. DEP analyzed the data at a county or state level. The EPA has analyzed the data on state and county levels as well.

The data used by EPA is from 1996. Since then, EPA has issued standards that affect many industry categories, such as chemical plants, oil refineries, and steel mills; as a result, EPA expects a reduction in air toxics emissions from these standards. EPA standards have also reduced air toxics emissions from cars and other mobile sources.

Comparing modeled concentrations against health-protective thresholds means relying on toxicity data, which also contain uncertainty. The study concentrations should, therefore, be considered values of potential concern, rather than representations of absolute threat to public health.

This analysis is limited to outdoor exposure. Potentially important sources of risks to public health, specifically, indoor sources, are beyond the scope of EPA's NATA program.

Are there emissions data more recent than 1996?

EPA will estimate air toxic emissions for 1999 as part of the National Emission Inventory program. This 1999 emissions inventory data will be used to create a 1999 NATA, which EPA is expected to complete and make available to the public in 2005.

How does DEP use the NATA?

DEP can use the NATA results to identify potentially important emission sources and pollutants, set priorities for future regulatory programs to address toxics and sources that may present the most significant threat to public health, and evaluate the effectiveness of state programs.

Predominant Sources of HAPs Over A Massachusetts AAL in More Than One County Based on EPA's 1996 NATA Data

Hazardous Air Pollutant	**Predominant Source Categories, Historical and Present**
Acetaldehyde	mobile sources, wood stoves, *coal refining*
Arsenic	wood preserving, incineration, combustion activities
Benzene	mobile emissions, combustion activities
Butadiene (1,3)	mobile emissions, rubber and plastic production
Carbon Tetrachloride	pesticides*, fluorocarbon production*, dry cleaning*, solvents
Chloroform	dry cleaning*, solvents*, perchloroethylene production, chlorination of water
Chromium	combustion activities, chrome plating, treatment of cooling tower water
Ethylene Dichloride	lead scavenger in gasoline*, *production of vinyl chloride*
Formaldehyde	mobile emissions, fuel combustion, production of other chemicals
Methylene Chloride	degreaser, chemical intermediate
Perchloroethylene	dry cleaning, chemical intermediate, degreaser

*Source categories where hazardous air pollutants are no longer in use.
Sources in *italics* are not found in Massachusetts.

Hazardous Air Pollutants that Exceed a MA AAL in More Than One County Based on EPA's 1996 NATA Data

County	Modeled Hazardous Air Pollutants of Concern
Barnstable	benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, ethylene dichloride, formaldehyde, methylene chloride, perchloroethylene
Berkshire	benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, ethylene dichloride, formaldehyde, methylene chloride, perchloroethylene
Bristol	acetaldehyde, arsenic, benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, ethylene dichloride, formaldehyde, methylene chloride, perchloroethylene
Dukes	benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, ethylene dichloride, formaldehyde, methylene chloride, perchloroethylene
Essex	acetaldehyde, arsenic, benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, ethylene dichloride, formaldehyde, methylene chloride, perchloroethylene
Franklin	benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, ethylene dichloride, formaldehyde, methylene chloride, perchloroethylene
Hampden	acetaldehyde, arsenic, benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, ethylene dichloride, formaldehyde, methylene chloride, perchloroethylene
Hampshire	benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, ethylene dichloride, formaldehyde, methylene chloride, perchloroethylene
Middlesex	acetaldehyde, arsenic, benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, ethylene dichloride, formaldehyde, methylene chloride, perchloroethylene
Nantucket	benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, formaldehyde, methylene chloride, perchloroethylene
Norfolk	acetaldehyde, arsenic, benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, ethylene dichloride, formaldehyde, methylene chloride, perchloroethylene
Plymouth	acetaldehyde, arsenic, benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, ethylene dichloride, formaldehyde, methylene chloride, perchloroethylene
Suffolk	acetaldehyde, arsenic, benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, ethylene dichloride, formaldehyde, methylene chloride, perchloroethylene
Worcester	benzene, (1,3) butadiene, carbon tetrachloride, chloroform, chromium, ethylene dichloride, formaldehyde, methylene chloride, perchloroethylene